

## OUR PRESENT CONCEPTION OF ESSENTIAL HYPERTENSION †

By ROY E. THOMAS \*

ANY condition, the etiology of which is in doubt, is always a live topic. Add to unknown etiology, a steadily increasing incidence, and a mortality almost equal to that of any infectious disease, not excepting tuberculosis, and we have everything necessary to stimulate the keen interest of all students of medicine. Since Sir Clifford Allbutt first described the malady which he then called hyperpiesis, the rank and file of the profession have gradually come to recognize this condition as a clinical entity. Articles appeared from time to time in the medical journals, and as the importance of the subject became increasingly evident numerous theories concerning etiology, reports of experimental work and recommendations for therapy, resulted in confusing, rather than clarifying, the conception of hypertension for the average reader.

It is my intention to discuss briefly: (1) the theories concerning the etiology of this disease which to my mind merit consideration; (2) its pathology, including the relation it bears to arteriosclerosis, heart failure and the condition known as chronic nephritis; (3) clinical features and management, particularly prophylaxis based on our latest conceptions of etiology.

**Etiology**—It is probable that blood pressure may be raised by (1) increased heart action; (2) increased quantity or viscosity of the blood; and (3) increased resistance in the peripheral blood vessels. As early as 1862, Bezold<sup>1</sup> showed that stimulation of the accelerator nerve increased blood pressure, not by causing tachycardia, but by exciting the vasomotor center. Numerous experimenters have demonstrated that increased heart rate does not increase blood pressure as long as the regulating mechanism of the vasomotor system is intact. Of greater significance than the heart rate is the increased amplitude of the heart beat. Such an increase might be the result of direct stimulation of the heart muscle by toxins or chemical substances circulating in the blood stream. Increased output of the heart is probably not a factor in causing high blood pressure because of the compensating mechanism just mentioned.

The theory that increased viscosity of the blood causes high blood pressure was advanced by Ewald<sup>2</sup>. In favor of such a view is the fact that in erythremia there is a greatly increased viscosity of the blood with a normal or slightly increased volume output, and in many cases the blood pressure is elevated. Also the pressure lowering action of iodine salts (if

such exists) is possibly due to their action in decreasing the viscosity of the blood.

Can increased volume of blood cause high blood pressure? Most physiologists say not and support their stand by animal experimentation.

According to Tigerstedt,<sup>3</sup> enormous quantities of blood have been transfused with slight or very transient increase in blood pressure. The plethora is cared for by transudation into the tissues or more likely by a vasomotor protective or regulating mechanism which, according to Fazzani,<sup>4</sup> must be located in the periphery, since it is effective after section of the spinal cord. So much for increased heart force and changes in the volume or composition of the blood. It is pretty generally accepted that they are at the most very minor and transient factors in the cause of increased blood pressure.

This leaves only increased resistance in the peripheral vessels as the chief etiological factor in the mechanics of hypertension. While students of the problem generally agree that increased peripheral resistance is the cause of high blood pressure there are many opinions as to just what causes this increased resistance; is it spasm, lessened elasticity, or pressure from without the vessel wall as in edema, or increased intra-abdominal pressure? Little can be said in favor of external pressure as a cause of increased peripheral resistance. No more conclusive is the evidence in favor of the mechanical theory, the advocates of which hold that a primary contraction of the kidney is the chief factor in causing peripheral resistance. If merely a local narrowing of the arterial tree could cause high blood pressure we should see it in all cases of Renaud's disease and endarteritis obliterans. Loss of elasticity in the vessel walls as the cause of high blood pressure can be dismissed with a word. The frequency of advanced generalized arteriosclerosis associated with normal or low blood pressure is well known. Kahler<sup>5</sup> believes that the greatest factor in the cause of high blood pressure is a general contraction of the precapillary arterioles, a disorder of the motor control of the blood vessel musculature. *This* is the theory which seems to fit in best with clinical observation and recent experimental work.

If we accept this theory of the mechanics of high blood pressure we have yet to explain the factors back of this loss of vasomotor equilibrium. To account for it many interesting hypotheses have been advanced, the most plausible of which are: (1) reflex—due to some peripheral, visceral or central stimulation, the latter possibly psychic in nature; (2) chemical—due to the presence in the blood of normal products of metabolism like guanadin or glucose in increased amounts as in hyperglycemia or abnormal products of metabolism or toxins reaching the circulation from the intestinal tract; (3) endocrine—through hyperactivity of the adrenals or pituitary glands or hypofunction of the gonads; (4) hereditary—either by direct transmission of some constitutional anomaly or special susceptibility to environmental factors.

It seems improbable that any one of these four hypotheses will explain all cases of chronic hypertension. Barker<sup>6</sup> has recently expressed his opinion

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as follows: "That the causes (of hypertension) be partly in the germ plasm and partly in environmental influences, seems certain. The tendency of essential hypertension to occur in families as well as its incidence among persons of certain types of constitutional make-up (those with vasopathic, neuropathic and endocrinopathic inferiorities) point to a predisposition that is geno-typically determined." As possible environmental factors, he mentions infectious processes, chronic intoxication, dietetic errors and exposure to stress and strain, mental or physical.

Heredity is without doubt the greatest single factor in the etiology of hypertension. Common habits and environment will hardly account for the high rate of incidence in certain families such as the one reported by Rosenbloom<sup>7</sup> in which both father and mother died of cerebral hemorrhage at forty-five and eight of ten children had marked hypertension before the age of fifty. The records of any medical clinic will furnish similar instances. O'Hare<sup>8</sup> has found a family history of vascular disease in 68 per cent of 300 cases of hypertension compared with 37.5 per cent in controls. Barach<sup>9</sup> found a positive history of cardiovascular disease in all but two of forty cases of chronic hypertension observed by him. He considers the factor of heredity the most prominent one in the hypertension group. He also considers acute infections a factor in as much as they bring about endocrine disturbances and neuro-circulatory asthenia. In an unselected series of 100 cases of chronic hypertension observed by the writer 65 per cent had a family history of cardiovascular disease. In an equal number of controls such a history was obtained in only 32 per cent.

In addition to certain physical characteristics (sthenic habitus), which seems to be prevalent in these families in which hypertension is prone to occur, I believe that there is a more or less characteristic mental make-up or disposition. These individuals are intense in everything they do. They worry without showing it. They cannot relax. If they play they do so with such concentration and effort that it really amounts to work.

As has been pointed out by Kylin<sup>10</sup> there are certain resemblances between hypertension and the disease of which bronchial asthma is a manifestation; both are hereditary, both occur in neurotic individuals with abnormally sensitive vegetative nervous systems, and both are characterized by spasms of involuntary muscle fibers—in the arterial wall in one case, and in the bronchial wall in the other.

*Pathology*—Fishberg<sup>11</sup> has described the arterial changes found in seventy-two cases of chronic hypertension which came to autopsy. Typical lesions occurred in the arterioles. These changes were most frequent and most marked in the kidney and consisted of: (1) hyalinization of afferent arteries beginning close to the junction with the glomerular tuft; (2) hyperplasia of the internal elastic membrane with reduplication and formation of multiple lamellae; (3) reactive proliferation of the neighboring connective tissue resulting in marked narrowing of the lumen which may go on to obliteration. The vessels of the spleen, liver, pancreas and brain are involved to less extent while those of the skin,

striated muscle, gastro-intestinal tract and heart apparently escape.

The gross changes which occur late in chronic hypertension are so well known as to require only brief mention. They consist chiefly of more marked renal changes, sclerosis of the larger arteries, cardiac hypertrophy and retinal or cerebral hemorrhages. It is upon these secondary changes that most of the symptoms of hypertension depend.

#### CLINICAL CONSIDERATION

Hypertension of comparatively short duration and obvious cause such as occurs in acute nephritis, toxemia of pregnancy, hyperthyroid states or as the result of intracranial lesions is not within the scope of this paper. In these conditions the hypertension is merely a symptom having no more significance than fever in infectious diseases. Kahler<sup>12</sup> has attempted an elaborate classification of chronic hypertension based upon pathogenesis, clinical manifestations and response to certain drugs, venesection and lumbar puncture. Inasmuch as many cases fail to conform to this classification it seems unwise to urge its general adoption lest it add to the confusion already existing.

The chief point under discussion at this time seems to be the relation of hypertension to chronic nephritis. Chronic nephritis may be divided into two types; one with edema, the other without edema (Christian.<sup>13</sup>) In the first type high blood pressure often occurs but is frequently absent. The glomeruli are chiefly affected and the disease usually may be traced to some infection. In the second type (without edema) high blood pressure is constant, the lesions in the kidney are chiefly vascular and proliferative, and no constant etiological factor has been demonstrated unless it be a pre-existing hypertension. Occasionally a case of clear cut hypertension is seen which can be followed through the gradual changes characterized by fixation of specific gravity of the urine, retention of sodium chloride and nitrogen until finally cerebral hemorrhage, heart failure or uremia end the picture. Who can deny that all cases of essential hypertension might not follow this course if they were not terminated by cardiac failure or cerebral hemorrhage before renal function became greatly affected?

The relation of hypertension to chronic heart disease is generally recognized and needs little comment. Janeway<sup>14</sup> has said that no large reduction of the mortality from circulatory diseases is likely until the problems of hypertension and rheumatism have been solved. In a series of 250 cases of myocardial insufficiency, he found the largest group (36 per cent) due to hypertension. In England and Scotland on the other hand the first place in the etiology of chronic heart disease is held by rheumatic fever (Cotton<sup>15</sup>). Allan<sup>16</sup> This seems significant when one considers the difference in temperament and mode of living between the English and American people.

Just how great a part chronic hypertension plays in the etiology of general arteriosclerosis is difficult to determine. Allbutt in his "Diseases of the Arteries including Angina Pectoris," mentions hyperpiesis first among the causes of arterial disease, and it is

safe to say that as our knowledge of the subject becomes more complete, we shall attribute to this cause still greater importance.

The manner in which hypertension causes anatomic changes has led to much speculation. Moschovitz<sup>17</sup> believes that many of them can be accounted for by a mechanical stretching of the tissues with replacement fibrosis followed by hyalinization and calcification. Thickening of the intima, hypertrophy of the media and increase in the elastic fibers he believes to be in a great measure compensatory.

The only constant manifestation of early essential hypertension is the symptom which has given to this disease its name, i. e., persistent high blood pressure unaccounted for by some evident cause such as hyperthyroidism, acute nephritis or toxemia of pregnancy. Of other early symptoms which occur the most common are fatigue, irritability, vertigo, insomnia, dyspnoea upon exertion, palpitation and digestive disturbances. Late symptoms depend upon impaired cardiac, renal or vascular function and are not in any way characteristic of hypertension.

It is not my intention to discuss in detail the management of hypertension. This phase of the subject has been well covered by Du Bray<sup>18</sup> in a paper read before this section in 1923. Dietetic restrictions, modified habits of work and play, sedative drugs, hydrotherapy, glandular extracts, venesection, etc., may all have their places in the treatment of this condition. The problem which confronts physicians today (a problem just as vital if less evident than that of cancer), is the prevention of hypertension. As physicians we could accomplish much by cultivating a closer relationship with the relatives and friends of the patients with whom we come in contact. We would then be in a position to advocate periodical physical examinations, shorter working hours under less tension and more frequent vacations of the right sort for the modern business man. We could preach the dangers of obesity and excessive use of tobacco. It might even be possible to influence the children in families prone to develop hypertension, to choose a life work in which they would be likely to meet a minimum of the stress and strain of life as it is lived in America today.

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#### THE APPENDICITIS PROBLEM †

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IT IS the duty of the section chairman to make an opening address before the group over which he presides. What he shall say is up to him. I have chosen to depart from the usual inspirational paper or yearly summary of progress to the section's credit and to sketch some phases of our problems as surgeons with the hope of stirring up some serious thinking. If we do not stop now and then and listen to those of our number who are strong for figures, we are too prone to remember only our successes and to forget our failures.

Willis in a recent paper on surgical mortality has served to confirm in the minds of some of us the fact that the mortality rate in appendicitis is entirely too high. Too frequently do we read in the public press that Mr. So-and-So died following an operation for appendicitis. Of course the newspaper never mentions the fact that Mr. Blank, who had never been vaccinated, died of smallpox. That would put the blame on the victim. The death following appendectomy is "good news stuff" because it is made to appear that if a meddlesome surgeon had kept his hands off the valuable citizen would still be alive. Humanity would benefit by the smallpox notice, the cause of public health be fostered. This is the situation we find ourselves in, quite a disconcerting one too, to say the least. I want to lay this matter of appendicitis and its mortality rate before you, as I have said, for thoughtful consideration.

Why is the mortality rate not decreasing, why is it indeed increasing, as the figures seem to indicate? It is a particularly serious question, for the fatalities are among the younger members of society, people at the beginning or at the height of their economic careers, together with a high proportion of children between the ages of 5 and 15. Some of these deaths can be laid at the feet of relatives and

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